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Smoking, Periodontitis and Inflammatory markers: Influence of scaling and root planing on NLR and PLR among smokers and nonsmokers.

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ABSTRACT

AIM:The aim of this study wasto estimate the neutrophil to lymphocyte ratio, Platelet to lymphocyte ratio in smokers with periodontitis and non-smokers with periodontitis before and after scaling and root planing.

Methods: A total of56 systemically healthy individuals, with stage I or II periodontitis, were divided into 2 groups: **Group 1**- 28 Nonsmokers with Stage I / II periodontitis. **Group 2**- 28 Smokers with Stage I / II periodontitis.Gingival index, Plaque index, Clinical attachment level, Probing pocket depth were assessed at baseline and 1 month after scaling and root planing. Laboratory parameters i.e. Neutrophil-to-lymphocyte ratio and platelet-to lymphocyte ratio was assessed by obtaining patients complete blood count and absolute lymphocyte count at baseline and 1 month after SRP.

Results: Statistically significant clinical improvements in plaque index, gingival index, clinical attachment level, and probing pocket depth were observed after scaling and root planning in nonsmokers and smokers. The neutrophil-to-lymphocyte ratio decreased after SRP in both smokers and non-smokers, but the difference was not statistically significant.Howeverplatelet-to-lymphocyte ratio values increased significantly after scaling and root planning in both groups.

Conclusion: SRP effectively improved periodontal health, by reducing plaque and gingival inflammation and changes in PPD and CAL. NLR values reduced after SRP in both groups although not statistically significant. PLR showed statistically significant changes. NLR may offer valuable insights into the association between periodontitis and systemic inflammation. However, the PLR may not serve as a systemic marker due to the influence of various factors such as gender, age, lifestyle traits, genetics, BMI, smoking, and environmental factors on PLR.

Keywords: Neutrophil-to-lymphocyte ratio; platelet-to lymphocyte ratio; Smokers; Non-smokers; scaling and root planing; Periodontitis.

INTRODUCTION

Periodontitis is an infectious disease that causes inflammation of the supporting structures of the teeth, leading to connective tissue attachment loss and alveolar bone loss.[1] Bacterial pathogens associated with various forms of periodontitis[2-4] can invade periodontal tissue and enter systemic circulation,[5,6] with the severity of gingival inflammation influencing the extent of bacteremia.[7,8] Inflammation, triggered by pathogens, damaged cells, and toxic substances,[9] initiates cellular and humoral changes, with white blood cell (WBC) count serving as a reliable hematologic marker for monitoring body inflammation.[10,11]

Neutrophils play a crucial role in periodontitis-related inflammation, responding to chemoattractant signals and migrating towards the gingival crevice, where oral microorganisms are present. They are found in both healthy and diseased periodontal tissues, with an increased influx during

disease states.[12] Other inflammatory cells, including lymphocytes and platelets, also contribute to the inflammatory response, with peripheral blood parameters like red blood cells, WBCs, and platelets being altered in periodontal diseases and linked to systemic inflammation.[13-15]

Neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are potential markers of systemic inflammation, reflecting the balance between innate and adaptive immune responses.[16,17] Smoking, a major risk factor for periodontal disease and systemic inflammation, exacerbates the release of inflammatory mediators into the circulation.[18-20] Given the systemic implications of periodontitis, analyzing markers such as neutrophils, NLR, platelets, and PLR can provide insights into the systemic and periodontal inflammatory response, particularly in smokers.[21-23] The aim of this study was to estimate NLR and PLR in smokers and non-smokers with periodontitis before and after scaling and root planing.

METHODOLOGY

A total of 56 individuals,between 20 and 65 years, were recruited for the study. Participants, including both cigarette smokers and nonsmokers with stage I and stage II periodontitis, were selected from the outpatients at the Department of Periodontology, Yenepoya Dental College, Mangalore. The sample size was calculated using G* Power software for an independent sample t-test at a 10% level of significance and 90% power, with a standard effect size of 0.8, resulting in a minimum of 28 participants per group, totaling 56.

The selected patients were divided into two groups:
Group 1: 28 nonsmokers with stage I or stage II periodontitis. Nonsmokers- adults who have never smoked or have smoked fewer than 100 cigarettes in their lifetime.

Group 2: 28 smokers with stage I or stage II periodontitis. Smokers were included based on the CDC classification for current smokers—adults who have smoked 100 cigarettes in their lifetime and currently smoke.

Stage I and stage II periodontitis were classified according to the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.
All patients received a verbal explanation of the study, and written informed consent was obtained. The study received clearance from the Institution's Ethical Committee.

Inclusion Criteria:

- Systemically healthy subjects with Periodontitis, aged 20-65 years from both genders.
- Non-smokers for group-1 and Current smoker in group-2.

Exclusion Criteria:

- Patients with systemic diseases and known allergies,Pregnant, lactating women, women in menopause,Patients with immunosuppressed conditions, Smokeless tobacco users, Patients who received periodontal therapy within the last 6 months,
- Antibiotic and/or anti-inflammatory drug regimen in last 3 months.

Clinical and laboratory assessments:
Participants from both groups were assessed for medical and dental history, and smoking status before periodontal examination. Theclinical parameters recorded at baselineand after 1 month after SRP included Gingival index[24], plaque index[25], probing pocket depth, and clinical attachment levelVenous blood was drawn from the antecubital fossa of the arm using a 21-gauge syringe into a vacutainer (Figure-1)todetermine complete blood count and NLR and PLR ratios.All participants underwent scaling and root planing using ultrasonic scalers and manual instruments, with local anesthesia when required. Oral hygiene instructions were provided and participants were recalled after one month for re-assessment of clinical and laboratory parameters.

LABORATORY PARAMETERS:

Calibrated automatic hematologyanalyzer (XN 1000, SysmexCorpn, Japan) (Figure-2) was used for the WBC and platelet estimation. Total neutrophil

count and total platelet count was obtained from patient's complete blood count.

Calculation of NLR and PLR

$$NLR = \frac{\text{Total neutrophil count}}{\text{Absolute lymphocyte count}}$$

$$PLR = \frac{\text{Total platelet count}}{\text{Absolute lymphocyte count}}$$

STATISTICAL ANALYSIS

Data gathered was analyzed using SPSS version 24.0.

1) Descriptive statistics
Mean and Standard deviation was used for continuous data frequency and percentage were used for categorical data and 95% confidence interval (CI).
2) Paired t-tests were conducted to assessintragroup comparison of clinical and laboratory parameters among smokers and nonsmokers, before and after SRP.
3)An independent t-test was conducted to assess intergroup comparison of clinical and laboratory parameters between smokers and non-smokers.
P value < 0.05 was considered to be statistically significant.

RESULTS

Age and genderdistribution:

The mean age in years of subjects in group 1 (n=28) was 44.64±10.40 and in group 2 (n=28) 45.11±13.12 (Table 1). Group 1 comprised of 12 male and 16 female participants and Group 2 comprised of 28 males.

Group 1- Non-smokers with periodontitis

The results showed significant reductions in all clinical parameters after 1 month following SRP. Specifically, the mean plaque index decreased from 1.64±0.91 at baseline to 0.20±0.14 after SRP. Likewise, the mean gingival index decreased from 2.28±1.56 to 0.72±0.58, the mean CAL decreased from 1.76±0.90 to 1.17±0.57, and the mean PPD decreased from 5.40±1.29 to 3.93±0.54. These improvements were statistically significant ($p < 0.0001$). (Table 2)

Group 2- Smokers with periodontitis

The results revealed significant reductions in all clinical parameters after 1 month following SRP. Specifically, the mean plaque index decreased from 1.79±1.22 at baseline to 0.32±0.12 after SRP. Similarly, the mean gingival index decreased from 2.00±1.19 to 0.46±0.40, the mean CAL decreased from 2.30±0.82 to 1.76±0.72, and the mean PPD decreased from 5.03±0.98 to 4.31±0.71. These improvements were statistically significant ($p < 0.0001$). (Table 2)

Intergroup comparison

Statistically significant differences were observed in all clinical parameters between the two groups.Non-smokers had a significantly lower plaque index compared to smokers after SRP.Smokers had a significantly lower gingival index compared to non-smokers after SRP. Non-smokers showed better improvement in CAL and PPD compared to smokers. (Table 3)

Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet- to- lymphocyte ratio (PLR)

Group 1- Non-smokers with periodontitis

The mean NLR at baseline was 2.17±0.95, which reduced to 2.05±0.77 after 1 month following SRP ($p=0.34$)indicating, no statistically significant change in NLR among non-smokers at baseline and 1 month after SRP. The mean PLR at baseline was 106.05±30.60 which increased to 125.90±57.79 1 month after SRP ($p=0.023$). Statistically significant increase in PLR after SRP among non-smokers was observed. (Table-4)

Group 2- Smokers with periodontitis

The mean NLR at baseline was 1.84±0.64, which slightly decreased to 1.78±0.63 after 1 month, indicating that the change in NLR was not statistically significant. ($p = 0.16$)The mean PLR at baseline was 84.94±24.72, which significantly increased to 100.00±39.33 after 1 month following SRP. There was a statistically significant increase in PLR after SRP. ($p = 0.005$) (Table-4)

Intergroup comparison

There is nostatistically significant difference between NLR among smokers and non-smokers. (p

≥ 0.157) Statistically significant difference was seen between PLR among smokers and non-smokers. Non-smokers had higher values compared to smokers. (p =0.055) (Table-5)

DISCUSSION

Periodontitis is a chronic inflammatory disease that significantly impacts oral health, function, and systemic well-being. It involves progressive loss of periodontal tissue integrity, marked by attachment loss and alveolar bone resorption, highlighting the severity of the condition and its broader health implications.[26]Traditionally, microbial biofilm accumulation at and below the gingival margin has been seen as the primary cause of periodontitis.[27]In patients with periodontitis, bacterial components or locally produced pro-inflammatory cytokines can enter the circulation, leading to systemic inflammation.[28]

Inflammation triggers immune system activation through the release of inflammatory mediators from periodontal tissues, leading to a systemic acute phase response.[29]Recent research[30] has delved into the systemic consequences of periodontitis, highlighting its association with various systemic conditions. Observational studies using quantitative analysis have consistently linked periodontitis to changes in WBC counts, particularly an increase in neutrophils and a decrease in erythrocyte and platelet lineages.[31,32] This suggests a systemic inflammatory response induced by periodontal infection.[33]

Periodontal bacteria can invade periodontal tissues, triggering a systemic response and releasing leukocytes into circulation. Chronic inflammation and bacterial interplay in periodontal tissues may stimulate the bone marrow to produce more inflammatory cells.[34]Recent research shows elevated levels of proinflammatory mediators such as NLR, PLR, WBC counts, C-reactive protein, neutrophil count, and thrombocyte count in individuals with periodontitis, linked to disease severity.[29,35-38]

Smoking is a well-established risk factor for periodontitis. The detrimental effects of smoking on periodontal tissues have been reported strongly through many researches.[39-41]Smoking, a well-known risk factor for periodontitis, exacerbates systemic inflammation through oxidative stress, endothelial injury, and platelet activation. Inflammatory biomarkers and platelet activation markers are elevated in smokers, contributing to arterial thrombosis and cardiovascular events.[40,42]Studies on smoking's effects on blood parameters, including NLR, PLR, and mean platelet volume,[28,43] with some reporting increased NLR and decreased PLR depending on smoking duration and intensity[44], and others finding no significant difference in NLR but a positive correlation with PLR.[23]

Non-surgical periodontal therapy aims to reduce or eliminate pathogens and metabolites to achieve optimal oral health and function with appropriate aesthetics, and ultimately to prevent the recurrence of periodontitis.[45]

Interventional evidence further supports that with nonsurgical periodontal treatment consistently reduces WBC counts and blood indices, indicating the potential therapeutic benefits of addressing periodontal inflammation.[35,36,37,45]Non-surgical periodontal therapy has also shown to improve the NLR and PLR in chronic periodontitis patients.[46]Further, a study by Christan C *et al*[15]found that smokers with generalised aggressive periodontitis had significant reduction in platelet count following periodontal therapy which may lead to decreased PLR.

The present study compared the NLR and PLR in smokers and non-smokers before and after SRP in periodontitis subjects. The clinical parameters, including plaque index, gingival index, CAL and PPD were assessed to evaluate the effectiveness of SRP in both groups and its effect on the blood parameters.

The clinical parameters in this present study showed statistically significant improvement in PI, GI, PPD and CAL among both non-smokers andsmokers with periodontitis following SRP. These findings suggest that SRP positively impacts clinical

parameters, aligning with a systematic review that highlighted SRPas an effective treatment for reducing probing pocket depth and improving the clinical attachment level.[47]

In this study, the investigation into the relationship between smoking and inflammatory markers, particularly NLR and PLR, revealed interesting findings. NLR values were higher in both smokers and non-smokers group at baseline which reduced after periodontal treatment but the reduction was not statistically significant in either intragroup or intergroup comparison. This aligns with study by Pujani*et al*.[23] wherein no statistical difference in NLR was seen between smokers and non-smokers without therapeutic intervention. This result may suggest that while periodontal therapy may improve oral health outcomes, it may not have a statistically significant impact on systemic inflammation.

It was observed in this study that the PLR values were lower in both the groups at baseline, with smokers exhibiting lower PLR compared to non-smokers. This finding is in accordance with the study conducted by Gummus*et al*.[44] which suggested higher NLR and lower PLR values in smokers indicative of thromboembolic risk among smokers. Mishra S *et al.*[48] observed a higher PLR among individuals with periodontitis compared to healthy individuals, although there was no statistically significant difference.

Research has shown that PLR values reduced after scaling and root planing.[46] However, an increase in the PLR was observed among both smokers and non-smokers 1 month after scaling and root planing in this study which was statistically significant. Various factors which influence the PLR ratios include: gender, age, lifestyle traits, genetics, body mass index, smoking and environmental factors as stated by Lin BD *et al*.[49]

Gender differences in PLR show higher levels in women due to higher platelet counts, influenced by lower serum iron levels and estrogen. Age also affects platelet counts, with younger individuals having higher counts and the elderly experiencing declines due to reduced hematopoietic stem cells. Seasonal variations, lifestyle factors like BMI affect NLR and PLR. Obesity and dietary habits also influence these ratios, linking higher BMI to chronic inflammation.[49]All these factors were not considered in this study, which could have influenced PLR values. Variations in PLR between non-smokers and smokers with periodontitis may be due to differing disease severity and gender distribution. Research shows that women have higher PLR and lower NLR compared to men.[49]

There are no universally accepted cut-off values for blood leukocyte ratios. However, observed changes in NLR and PLR remained within normal ranges: 0.43-2.75 for NLR in males, 0.37-2.87 for NLR in females, 36.63-149.13 for PLR in males, and 43.36-172.68 for PLR in females, according to a study in Chinese populations.[50]The NLR and PLR values in the present study are higher compared to other Indian studies, which reported a mean NLR of 1.9 and a mean PLR of 91.77.[46,48,51] This discrepancy could be attributed to regional differences.

The findings in the present study are in partial agreement with Loos *et al*.[52]who indicated a decrease in the number of leukocytes after non-surgical periodontal therapy in patients with generalised aggressive periodontitis. Conversely, smokers exhibited significantly higher PLR values, with a weak yet significant positive correlation observed between PLR and increasing pack-years of smoking.Acharya *et al*.[46]and Verma *et al*.[53]explored NLR and PLR in chronic periodontitis patients before and after nonsurgical therapy and concluded that these ratios could serve as potential biomarkers of systemic inflammatory response to periodontitis, aiding in understanding the link between periodontal health and systemic conditions.

The limitations of this study include a small sample size.Gender distribution among the groups, age, lifestyle traits, genetics, body mass index, regional variations and environmental factors that are implicated in affecting these biomarkers was not considered in this study.

Based on the findings of this study, SRP as a treatment for periodontitis resulted in significant

improvement in various clinical parameters thereby reducing local inflammation and improving the periodontal health in both smokers and non-smokers. The study also provides insights into the hematological parameters, specifically the NLR and PLR, in smokers and non-smokers undergoing SRP. NLR values exhibited a reduction post-treatment though not statistically significant between the two groups. The PLR values increased after scaling and root planing and was statistically significant.

CONCLUSION

Scaling and root planing improved clinical parameters and reduced local inflammation in both smokers and non-smokers with periodontitis. No statistically significant changes were observed in NLR among smokers and non-smokers. However, there was statistically significant difference in PLR. NLR may offer valuable insights into the association between periodontitis and systemic inflammation. However, PLR may not serve as a systemic marker due to the influence of various factors such as gender, age, lifestyle traits, genetics, body mass index, smoking, and environmental factors on PLR. Further research is required to elucidate the specific mechanisms underlying the observed changes in inflammatory markers.

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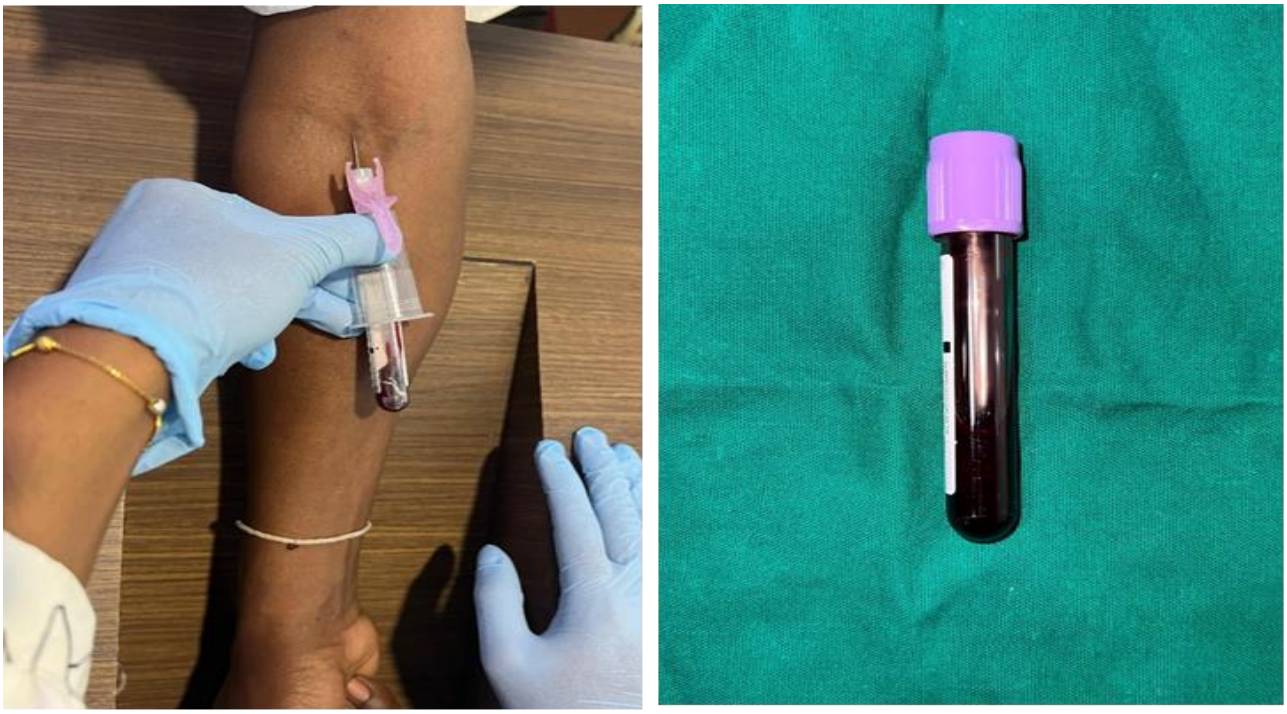

FIGURE 1: COLLECTION OF VENOUS BLOOD FROM THE ANTECUBITAL FOSSA OF THE ARM USING A 21 GAUGE SYRINGE

FIGURE 2: HEMATOLOGY ANALYZER (Sysmex XN-1000™)

TABLES:

Table 1: Age in years of group 1 and group 2

	GROUP	Minimum	Maximum	Mean Standard Deviation
AGE	NS	21	66	44.64±10.40
	S	20	63	45.11±13.12

Table 2: **Comparison of clinical parameters in group-1 and group 2 before and after SRP**

CLINICAL PARAMETERS		Mean Standard Deviation		p-value
		Group-1	Group-2	
PLAQUE INDEX	BASELINE	1.64±0.91	1.79±1.22	< 0.0001
	1 MONTH	0.20±0.14	0.32±0.12	
GINGIVAL INDEX	BASELINE	2.28±1.56	2.00±1.19	
	1 MONTH	0.72±0.58	0.46±0.40	
CLINICAL ATTACHMENT LEVEL	BASELINE	1.76±0.90	2.30±0.82	
	1 MONTH	1.17±0.57	1.76±0.72	
PROBING POCKET DEPTH	BASELINE	5.40±1.29	5.03±0.98	
	1 MONTH	3.93±0.54	4.31±0.71	

Table 3: Comparison of clinical parameters between smokers and non-smokers at baseline and 1 month after SRP

INTERGROUP COMPARISON		Group	Mean Standard Deviation	p-value
PLAQUE INDEX	Baseline	Non-smokers	1.64±0.91	0.61
		Smokers	1.79±1.22	
	1 month	Non-smokers	0.20±0.14	0.001
		Smokers	0.32±0.12	
GINGIVAL INDEX	Baseline	Non-smokers	2.28±1.56	0.45
		Smokers	2.00±1.19	
	1 month	Non-smokers	0.72±0.58	0.057
		Smokers	0.46±0.40	
CLINICAL ATTACHMENT LEVELS	Baseline	Non-smokers	1.76±0.90	0.025
		Smokers	2.30±0.82	
	1 month	Non-smokers	1.17±0.57	0.001
		Smokers	1.76±0.72	
PROBING POCKET DEPTH	Baseline	Non-smokers	5.40±1.29	0.023
		Smokers	5.03±0.98	
	1 month	Non-smokers	3.93±0.54	0.031
		Smokers	4.31±0.71	

Table 4: Comparison of Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet- to -Lymphocyte ratio before and after scaling and root planing (SRP)

LABORATORY PARAMETERS		Mean Standard Deviation	
		Group-1	Group-2
NLR	BASELINE	2.17±0.95	1.84±0.64
	1 MONTH	2.05±0.77	1.78±0.63
	p-value	0.340	0.160
PLR	BASELINE	106.05±30.60	84.94±24.72
	1 MONTH	125.90±57.79	100.00±39.33
	p-value	0.023	0.005

Table 5: Comparison of Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet- to -Lymphocyte ratio (PLR)between Smokers and Non-smokers at baseline and 1 month after SRP

INTERGROUP COMPARISON		Group	Mean Standard Deviation	p-value
NLR	Baseline	Non-smokers	2.17±0.95	0.14
		Smokers	1.84±0.64	
	1 month	Non-smokers	2.05±0.77	0.15
		Smokers	1.78±0.63	
PLR	Baseline	Non-smokers	106.05±30.60	0.006
		Smokers	84.94±24.72	
	1 month	Non-smokers	125.90±57.79	0.055
		Smokers	100.00±39.33	
		Smokers	106.05±30.60	